

additional perforating veins may be present as indicated by tests done previous to surgery. However, geniculate tributaries are an almost constant finding in this area, as well as below the knee. Because it creates a bifurcation, any sizable tributary will offer resistance to the Mayo stripper and this in turn will serve as a guide as to where additional small linear incisions should be made. At each point of bifurcation, the offending tributary may either be ligated or divided, or if its size warrants, it too may be followed distally either through digital dissection or use of the stripper.

Essentially the same basic pattern may be followed in the leg where previous tests serve as a guide to possible points where perforating veins may be present. Small exploratory incisions may also be made in these areas, and the offending vessels traced to their point of entry into the deep fascia, where they are tied and divided; the remaining fascial aperture is then closed with appropriate sutures. Not infrequently the site of a tributary will coincide with, or approximate the level of a perforating vein. As a consequence, the total number of incisions should at no time be excessive. At any rate, the saphenous dissection is carried to a point below the lowest perforating vein, where it is divided. All wounds are closed with interrupted mattress dermal.

Whenever the short saphenous is obviously dilated or incompetent as revealed by appropriate tests, the patient is turned and a transverse incision is made on the lateral aspect of the popliteal fossa. The fascial sheath is incised sufficiently to give adequate exposure of the underlying structures, the short saphenous vein is identified and ligated at its point of juncture with the popliteal vein. Care should be exercised not to injure the tibial nerve which lies immediately lateral to the popliteal vein. If indicated, the short saphenous is then followed distally in the same manner described for the internal saphenous; any perforating veins encountered, particularly in the mid-calf region, should be followed to their approximate point of juncture with the deep system. The fascial incision in the popliteal fossa is then closed with interrupted black silk. The skin closure is made as previously described. To allay any subsequent bleeding, firm pressure bandages are then applied from the toes to the groin.

The procedure advocated requires meticulous and painstaking surgery and the operation is not a short one, but when carefully done there is no attendant shock and the mortality is infinitely lower than any other surgical procedure of comparable duration. Regardless of the extensiveness of the surgical procedure, early ambulation is a mandatory order, and only under extenuating circumstances is the patient allowed to remain in bed longer than twenty-four hours after surgery. Should the latter be necessary, active and passive leg exercises are instituted to combat venous stagnation. Patients usually leave the hospital the first postoperative day with a minimum of discomfort. Any postoperative edema, attributable to circulatory and lymphatic readjustment, is transient, and can be readily controlled with firm elastic bandages.

Attempts at complete removal of the saphenous vein were made many years ago. However, the reasons the procedure initially fell into disrepute have been currently largely eliminated. When it was originally introduced, no preoperative sclerosing was done to limit surgical bleeding and obviate postoperative hemorrhage, and the dissection of the sapheno-femoral junction was undoubtedly not complete in many instances. Then too, the importance of the perforating veins was not appreciated and these were merely torn in removal of the saphenous stem, thus leaving intact, particularly in cases where the inner saphenous stem was undisturbed, direct as well as indirect connections between the deep and superficial systems. An unnecessarily high percentage of recurrence

naturally resulted. In addition, patients were kept in bed following surgery for lengthy periods, a fact which materially contributed to postoperative complications. All these factors played an important part in discrediting what was really a material advance in varicose vein therapy.

CONCLUSION

The surgical treatment outlined is more extensive than its predecessors, but any more conservative approach at least fails to eliminate all currently demonstrable etiologic factors. Regardless of the type of therapy, under no circumstances can the incidence of residual or recurrent pathology be considered negligible, and surgical results are still influenced by the extent of the varicosities at the time of surgery. Manifestly a much better result can be anticipated when the pathology is limited in both extent and severity. At any rate, those who devote themselves to the problem will find venous pathology of the lower extremities a most interesting field in which much constructive work is yet to be done.

The above procedure is suggested as a means of obtaining a more satisfactory end result in the treatment of varicose veins.

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RHEUMATIC FEVER: ITS RECOGNITION*

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I.—Introductory Statement.—The importance of the topic to be discussed is evidenced by the facts that 2 per cent of the school children of the United States have heart disease as a result of the rheumatic state; that 98 per cent of all heart disease in patients under 20 years of age is of rheumatic fever etiology; that it causes more deaths in the first two decades of life than all of the communicable diseases plus pneumonia; that it causes annually ten times more deaths than infantile paralysis; and that a study of the young men rejected for military service because of heart disease showed the rheumatic state to be the etiologic factor in 51 per cent in Boston, 70.3 per cent in Chicago, 64.4 per cent in New York, 65.9 per cent in Philadelphia, and 39.6 per cent in San Francisco. (White, Levy & Stroud; J.A.M.A., Dec., 1943.) The importance is further emphasized by the recent knowledge that rheumatic fever is not a disease confined to the temperate zone but that it is almost as prevalent in subtropical climates as in the temperate climates (Sampson; *Amer. Heart*, Feb., 1945) and that even the acute fulminating type of the disease is frequently seen in the tropics. (Huntington; Personal Communication, 1945.) The belief that the disease is confined to the temperate zone is due to the more frequent occurrence of the severe types in that zone.

This important disease now becomes a global problem. Hereditary susceptibility to the disease, previously thought to be a Mendelian recessive character (Armstrong and Wheatley; *Metropolitan Life Insurance Co.*, Nov., 1944)

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may be largely due to poor nutrition or to group crowding as shown by the experience of the Army and Navy in training centers. Good nutrition and segregation may be a large factor in protecting the individual from an inciting agent such as the Beta streptococcus hemolyticus (Wilson; Rheumatic Fever, Commonwealth Fund, 1940). It is true that children of parents who have had rheumatic fever are more susceptible to the development of the disease, in the presence of a streptococcus hemolyticus infection, i.e., tonsillitis, scarlet fever and otitis media, than are those children of non-rheumatic parents. (Paul; Epidemiology of Rheumatic Fever, 1943.)

DEFINITION

Out of the combined experience of the clinician, pathologist, bacteriologist, allergist, neuropsychiatrist and dermatologist working with this disease in a very large group of patients of military age, a consensus of opinion has gradually developed that we are dealing with a disease entity best characterized by the following definition: *Rheumatic Fever is a systemic anaphylactic inflammatory disease with protean manifestations of varying severity and duration.* Pathologic, histologic and clinical observation shows that the disease is a hypersensitivity angitis manifest in all of the body structures from the skin to the smallest subdivision of the viscera.

THE PHASES OF THE RHEUMATIC STATE

The *first phase* of the disease is that period of illness during which the patient is the host to the beta type of the hemolytic streptococcus. From military experience it has been learned that about 5 per cent of trainees develop some manifestation of a hemolytic streptococcus upper respiratory infection. This upper respiratory infection may take the form of a pharyngitis, tonsillitis, otitis media or a sore throat with a scarlatiniform rash, the so-called scarlet fever. Approximately 5 per cent of this group of patients develop the symptoms and signs of rheumatic fever. Therefore, it is logical to consider every patient with a hemolytic streptococcus throat infection a potential rheumatic fever subject. In those susceptible individuals who develop rheumatic fever it is proper to consider this the first-phase of the disease.

The *second phase* is the period of latency lasting from one to four weeks in duration. It is during this, the second phase of the disease, that the patient develops a hypersensitivity, is found to tire easily, may run a mild fever and have a persistent leucocytosis, elevated blood sedimentation rate and a gradual increase in the level of the antistreptolysin titre. (Rautz; Paper read before the California Heart Association, 1944.) This second phase is frequently noted only as an interval of time wherein the painstaking physician will find that the patient is not symptom free.

The *third phase* is the well-known period of rheumatic fever activity. The protean manifestations may be grouped into four distinct types, each of which with its symptom complex will be described in detail subsequently. The severity and duration of the phase of activity cannot be predicted as the most fulminating onset may be followed by a relatively short period of activity, for example, from four to six months, while in the sub-clinical type activity may persist for years.

The *fourth phase* is the period of rheumatic fever inactivity. It is the period of the arrested rheumatic state, or the period of rheumatic quiescence. This phase of the disease may be long or short, depending upon reactivation of the rheumatic state. The mild pain occasioned by muscle jelling, joint stiffness and soreness resulting from fatigue or exposure do not constitute a reactivation of the rheumatic process. An upper respiratory infection followed after a short interval of fever, tachycardia,

malaise, dyspnea, migrating polyarthritis with concomitant laboratory findings of an increased blood sedimentation rate, leucocytosis and electrocardiographic changes constitute a reactivation. Of a large number of patients returned to a rheumatic fever unit for supposed reactivation, only 2.4 per cent were found to have a reactivation. Thus, the fourth phase of the disease begins with the termination of rheumatic fever activity and ends only if reactivation occurs.

ONSET OF THE RHEUMATIC STATE

The beginning of the active stage of rheumatic fever may be insidious and go unnoticed and undiagnosed for a long period of time, as shown by the survey of rejectees with cardiovascular disease. (Levy, Stroud and White; J.A.M.A., Dec., 1943). It was found that 71.2 per cent of patients in four cities and 50 per cent in two cities did not give a history of the acute rheumatic fever. On the other hand, the onset of the rheumatic state may be very severe. The younger the patient, the more severe the onset, appears to be the rule. The latency phase of the disease is ended by an acute illness characterized by migrating polyarthritis, fever, shortness of breath, tachycardia out of proportion to the elevated temperature, loss of appetite and severe sweats. The patient is restless but moves the extremities very little because of painful joints. The face is flushed. The tongue and lips are dry. The skin is hot and dry alternating with periods of drenching sweat. The migrating polyarthritis usually begins in the larger joints such as the knees, ankles, elbows, wrists and at times the hip and shoulder joints. The periarticular tissues are swollen, hot and tender. As the process begins to subside in one joint, it lights upon an analogous joint. The polyarthritis persists a relatively short time after the patient has been put to rest. The administration of salicylates sufficient to raise the blood level to 30 milligrams per 100 cc. of blood relieves the polyarthritis promptly. Those patients with sedentary duties do not develop the severe joint manifestations that characterizes the physically active group.

The fever may be absent, moderate or severe. The severely elevated temperature persists for 10 to 18 days and then gradually subsides or it may persist at a low level for months.

The shortness of breath is usually overlooked. It is very common in the acute stage and is the first sign of myocardial failure and/or rheumatic pneumonitis.

The tachycardia is out of proportion to the degree of fever. Even though fever is absent, a tachycardia is usually present. The tachycardia persists as long as there is activity of the disease.

During the acute onset, carditis is shown by dilation, gallop rhythm and electrocardiographic changes may present itself. Pericarditis as indicated by a to-and-fro friction rub or by an effusion, is not frequently diagnosed. In our series of patients it was diagnosed clinically in 2.82 per cent of cases. Endocarditis is diagnosed only if the murmur is: (1) diastolic in time; (2) if a systolic murmur persists through six months' observation; (3) if the systolic murmur remains the same or grows worse in intensity and is heard in all positions and in all phases of respiration; (4) if the systolic murmur is transmitted; and (5) if the chamber enlargement or systolic thrill are found in the later period of the six months' observation. These signs of carditis may be present early in the acute onset but most frequently are later manifestations.

Pneumonitis was the presenting factor in 12 per cent of two hundred patients with prolonged rheumatic fever. The symptoms of rheumatic pneumonitis in order of their frequency appear to be, shortness of breath, tachycardia, fever, pleuritic pain, malaise, and cough. The shortness of breath and tachycardia may precede the fever, pleurisy,

and cough by many hours to a few days. The physical findings are those of small areas of dullness and diminished breath sounds anywhere in the lung fields. Fine râles soon appear. The patchy areas of pneumonitis shift from one site to another. The upper lobes are involved as frequently as the lower lobes. A pleural effusion may appear very rapidly. It disappears with equal rapidity unless congestive failure intervenes. Thorocentesis, like pericardial drainage, is seldom necessary. The x-ray findings of rheumatic pneumonitis are those of so-called primary atypical pneumonia. The pathologic histology is that of a pulmonary angitis with multiple small infarctions.

Weight loss and loss of appetite are very common at the onset. Returning appetite and gain in weight are good indices that the rheumatic activity is subsiding.

Abdominal pain is due to localized peritoneal irritation resulting from the angitis or capillary involvement in the parietal and visceral peritoneum. Confusion with acute appendicitis is not frequent. In the acute onset of rheumatic fever, abdominal pain is frequently due to diaphragmatic pleurisy or to peritoneal irritation. Usually the dyspnea, tachycardia, signs of pneumonitis or carditis, and polyarthritis are out of proportion to the abdominal pain. If the abdominal pain localizes and the classic picture of acute appendicitis persists, then operation should be done. In our series as will be reported in another paper, the diagnosis was not difficult. The problem of acute appendicitis at the onset of acute rheumatic fever was not frequently encountered. The patients who developed acute appendicitis did so during the prolonged convalescence and the number did not exceed the expectancy in a similar age group of non-rheumatic fever patients.

Chorea at the onset of acute rheumatic fever is very rare in our patients. Only .04 per cent of rheumatic fever patients developed chorea at any time during the period of observation.

At the onset of the disease, erythemas are frequent. The erythemas are those of an anaphylactic nature, varying from erythema marginatum to urticaria. Erythema multiforme and erythema nodosum are not manifestations of rheumatic fever activity.

Subcutaneous nodules are not frequently seen at the onset in patients of military age. Subcutaneous nodules when present, do not necessarily indicate a severe rheumatic activity.

Epistaxis is quite common at the onset of the disease in children. In the age group from 18 to 25 years 0.3 per cent patients show this phenomenon. However, in another study (Montgomery, 1945) it has been shown that there is a very definite increase in capillary permeability and fragility in the rheumatic state.

Concomitant with the onset of the disease there is an increase in the leucocyte count and an elevation of the blood sedimentation rate and of the antistreptolysin titre. None of these findings are specific for the rheumatic state.

THE CLINICAL TYPES OF RHEUMATIC FEVER

There are four clinical types of rheumatic fever recognizable, namely, the acute fulminating, the subacute polycyclic, the subacute monocyclic and the subclinical.

The Acute Fulminating Type was found to occur at a rate of 4.3 per cent in a series of patients who gave evidence of rheumatic activity for more than six months. This type is more frequent in childhood. The acute fulminating type is characterized by a sudden onset with migrating polyarthritis, fever, restlessness, severe tachycardia, dyspnea and drenching sweats. Carditis appears rapidly as shown by dilatation, gallop rhythm, murmurs, and at times pericarditis. Pneumonitis is present in many patients. Pneumonitis can be detected by careful physical examination, but is more frequently diagnosed by the

x-ray studies of the lungs. The migrating polyarthritis is of short duration although usually severe at the onset. The fever is relatively high, ranging from 101 to 103 degrees. The acute febrile period lasts from 10 to 18 days and then subsides with a rapid convalescence or goes into a prolonged low-grade state of activity. The tachycardia is marked and is the last sign to disappear. The leucocyte count is moderately elevated with a marked increase in the granulocytes. The urine may contain albumen and a moderate number of cells. Approximately 20 per cent of patients in this group show electrocardiographic changes indicative of myocardial and pericardial involvement. The acute fulminating type usually terminates with one cycle. This group represents those patients who are most susceptible to the disease. Although the mortality is low in the primary attack, due to a marked hypersusceptibility, these patients are more likely to have fatal reactivations.

The Subacute Polycyclic Type is similar to the acute fulminating during the onset except that it is less severe. It is more frequent. The percentage of occurrence in our series of patients was 27.6 per cent. The cycles may be short and spaced at long intervals, or in the more severe subtypes the cycles may be long and spaced at shorter intervals of time. It is in this type that the mortality is the greatest. Concerning fatalities 63.6 per cent in our series of patients were in this group. Likewise in this group with repeated cycles of activity, the most marked cardiac enlargement, most frequent signs of endocarditis and pericarditis are found. During one or all of the cycles pneumonitis of greater or less degree occurs. After the first cycle, polyarthritis is usually absent, and the response to salicylate therapy is poor. Congestive heart failure, complicating carditis occurs almost exclusively in this group of cases.

The Subacute Monocyclic Group is the most frequent of the four types occurring in our series in 61.3 per cent. The onset may be very mild and scarcely recognizable, or it may be moderately severe with moderate fever, mild migrating pains, a definite tachycardia and moderate elevation of the leucocyte count and blood sedimentation rate. The response to salicylates is usually prompt and the acute onset lasts for seven to fifteen days. Thereafter the course may be that of a rapid return to the stage of quiescence so that full activity may be resumed in from four to six months; or the course may be long with signs of mild activity persisting over a period of six to nine or more months.

The Subclinical Type was found in 5 per cent of our series. This group comprised those patients who may or may not have had an upper respiratory infection, and who in the course of a check up examination were found to have developed definite signs of rheumatic heart disease. Such signs are those of cardiac chamber enlargement, mitral and/or aortic insufficiency murmurs, paroxysmal tachycardias and auricular fibrillation. Upon careful observation this group of patients exhibited a few signs of rheumatic activity such as a cyclic low grade temperature, and a mildly increased sedimentation rate. At other times no confirmatory evidence of rheumatic activity was found. Two patients within this group died suddenly. Death in each instance was due to an acute anaphylactic reaction with the pouring out of collagen into the walls of the coronary arteries sufficient to cause an occlusion and myocardial infarction.

THE CRITERIA FOR THE DIAGNOSIS OF RHEUMATIC FEVER

The diagnostic criteria have been previously laid down by Swift, H. E. (J.A.M.A., June, 1929), and by Jones, D. E., (Stroud; Cardiovascular Disease, 1943).

The diagnostic criteria are subdivided into major and minor groups:

The major criteria are five in number, namely, a history of a previous attack, carditis, fever, migrating acute arthritis and chorea;

The minor criteria in order of their relative importance are, pneumonitis, tachycardia, subcutaneous nodules, erythemas, epistaxis and purpura, weight loss and abdominal pain. Iritis and conjunctivitis are too infrequent and indefinite to be included.

A previous history of rheumatic fever was obtained in 28.1 per cent of our patients. The importance of a previous history is of great value. Too often, however, mild arthralgia and muscle jelling are mistaken for a previous history. A previous history is of great diagnostic helpfulness when a patient presents himself with one or more of the major criteria and two or more of the minor criteria.

MAJOR DIAGNOSTIC CRITERIA

Carditis is probably the most important of the major diagnostic criteria. Carditis can be said to be present when a tachycardia, especially a resting pulse of 100 or more, persists; when there is dilatation of the heart as shown by enlargement and/or gallop rhythm; when pericarditis is evidenced by a to-and-fro pericardial friction rub and/or a pericardial effusion; when myocarditis is demonstrated by dilatation, gallop rhythm and electrocardiographic changes; and when endocarditis is established by hearing definite organic cardiac murmurs.

Fever is considered to be the most frequent evidence of an active rheumatic state. It is the most reliable evidence of activity in the absence of demonstrable carditis. At the onset the temperature range may be normal or as in the more severe types as high as 102 to 104 degrees. The degree of fever is not the paramount observation, but persistence of the fever is of utmost importance. No clinician can be sure that activity has ceased unless a carefully taken and recorded temperature curve has been observed for at least six to eight weeks time. Persistent fever in the subacute types of the disease takes one of two forms. The more frequent form is an evening rise to 99 to 100 degrees. The more insidious and most frequently unobserved form is a cyclic type of curve. In this form the temperature remains at a normal or a slightly subnormal level for one to two weeks and then becomes mildly elevated for a few days or as long as a week. It is rare for fever to be the only evidence of activity. Persistent fever must be viewed seriously especially if two or more minor criteria are present.

Polyarthritis, by which term is meant migrating acute arthritis, for over three centuries has been considered the significant sign of the disease. In recent years the literature has emphasized the unimportance of the joint signs. In the primary attack the joint signs are usually prominent and frequently the presenting symptom and sign. The major joints become red, hot, and swollen in a migrating fashion. With rest, the joint signs disappear quickly. The salicylates in adequate dosage are specific and prompt in action. The red, hot swollen joints subside leaving no residual pathology in the joints of the younger age groups. In those patients in the third decade of life arthralgia may persist. The persistent arthralgia is not considered a sign of activity, when all other criteria have disappeared. If one major or two minor criteria persist, then the complaint of painful joints must be considered evidence of activity.

Chorea is a relatively rare condition in the second and third decades of life. It has occurred in our series of patients in .047 per cent. None of the cases have exhibited the severe choreiform movements seen in childhood. There have been other neurological manifestations occurring during the active course of the disease. Two patients developed temporary hemiplegia with gradual complete recovery. Six patients developed unilateral or

bilateral brachial plexus involvement varying from weakness and pain in the entire upper extremity to wrist drop. Two patients developed temporary foot drop. These neurologic signs were not considered of psychic origin. With continued treatment of the rheumatic state the organic neurogenic signs cleared up.

MINOR DIAGNOSTIC CRITERIA

The minor criteria in the order of their relative importance are herewith described.

Pneumonitis has been overlooked by most American clinicians until: Baas, C. P. and Schwartz, S. P. (Am. Ht. J., April, 1927); Paul, J. R.: (Medicine, December, 1928); Swift, H. F. (J.A.M.A., June, 1929); Curnburn, A. F. "The factor of Infection in the Rheumatic State," 1931; and Eiman, John (Am. J. Med. Sc., March, 1932)—called attention to the clinical and pathologic findings. The constant occurrence of pneumonitis in the acutely ill and the frequency with which it is found in the very mild stages of activity emphasizes its importance. Frequently the chief complaint of the patient is dyspnoea and the first outstanding sign is that of pneumonitis, likewise, it is frequently the first sign found in a current cycle and in a reactivation. When the x-ray picture of the lungs is that similar to an atypical pneumonia and the sputum yields no specific organism in a rheumatic fever susceptible patient, the diagnosis of rheumatic pneumonitis is considered established.

Tachycardia is a minor diagnostic criteria which if constant and persistent during sleep is of positive value. Paroxysmal tachycardia and auricular fibrillation are rare in our series of patients.

Subcutaneous Nodules are not seen frequently. These are a definite sign of activity. In the past their significance has been considered ominous, but perhaps that is due to the fact that they are not looked for carefully in the subacute and mild cases. They are a diagnostic sign but do not carry the poor prognosis formerly attributed to them.

The Purpuras and Epistaxis. There is a definite increased fragility and permeability of the capillaries in the rheumatic subject as is shown by one of our co-workers, Leut. Comdr. Hugh Montgomery. The purpuras are seen rarely. Not over .3 per cent in our entire case load have shown purpura. Epistaxis is of similar significance and occurs very infrequently in the 2nd and 3rd decades. When purpura is present it is a very helpful diagnostic sign.

Weight Loss is a very common occurrence in the acute phases of the active state and weight gain is commensurate with the improvement in the patient.

Abdominal Pain has been considered of great importance in the literature. It is a relatively uncommon occurrence late in the disease. At the onset of an acute episode there may be abdominal pain without definite localizing signs but if there is a previous history of rheumatic fever and the findings of shortness of breath and tachycardia out of proportion to the fever, and especially if there are joint manifestations, it is safe to wait and watch, but if in doubt, then operate as the risk is small.

Recurrent Cycles may be initiated by surgery such as dental extractions or tonsillectomy. The fracture of bones is almost sure to set up a recurrent cycle of activity. Therefore it is unwise to do dental extractions or to perform tonsillectomy until six months have elapsed after activity has ceased. Fractured bones must be firmly fixed as soon after fracture as possible.

Laboratory Findings are not specific in rheumatic fever. There is no specific laboratory diagnostic test.

Anemia of the secondary type may develop rapidly in the very acute types. In the mild types of the disease anemia is uncommon.

Leucocytosis. The white blood count during the acute onset may be as high as 15,000 to 24,000 with as much as 90 to 95 per cent polymorphonuclear leucocytes. In the subsiding stage the leucocyte count falls rapidly to normal and is not a reliable guide as to the degree of activity.

Anti-Streptolysin Titre is elevated at the onset and continues to remain elevated for a long period of time. Because of the technical difficulties and because it remains elevated after other signs of activity have disappeared, it is not a satisfactory guide as to the degree of activity or guide in treatment. Its greatest value is in the epidemiology of rheumatic fever.

Blood Sedimentation Rate is elevated during the time of the phase of activity. It is the best single guide to the degree of activity. If the sedimentation rate remains elevated when all other signs of activity have disappeared, then the clinician must rule out other causes for an increased sedimentation rate.

Electrocardiographic Changes indicative of rheumatic activity are three—first a shifting A.V. conduction time, especially if the P.R. interval is well over .24 seconds. This change is frequently seen in varying degrees up to the Wenckebach phenomena and complete auriculo-ventricular dissociation; second, the typical electrocardiographic picture of pericarditis and third, the Q.R.S. and T. wave changes which are seen in myocardial damage resulting from the anoxia of coronary insufficiency. The coronary insufficiency is the direct result of arteritis of moderate to severe degree.

Urinary Changes in our experience have been minor and notable for their absence. In the acute phases albumin and a moderate number of cells may appear.

THE DIFFERENTIAL DIAGNOSIS

Rheumatic Fever in its acute form must be differentiated from Dengue Fever, Undulant Fever, Rheumatoid Arthritis, Chronic Osteoarthritis and Tuberculosis. This is best done by evaluating the above outlined criteria and by close observance of the clinical course.

In Dengue Fever the course is short, and there is no carditis or prolonged elevation of the sedimentation rate or the antistreptolysin titre.

Undulant Fever is ruled out by a persistently negative cross agglutination test with the *Bacillus Abortus* and *Bacillus Melletensis* and by the failure to develop carditis.

Rheumatoid Arthritis is difficult to differentiate early, but time and visual joint changes, and x-ray joint changes together with a low antistreptolysin titre point to this entity rather than rheumatic fever.

In Osteoarthritis, the development of x-ray evidence of joint pathology and the lack of most of the major and minor criteria of rheumatic fever serve to make the differentiation early.

Tuberculosis is confused by the prolonged fever, but here again the absence of the other four major criteria and most of the minor criteria rules out rheumatic fever.

SUMMARY

The recognition of rheumatic fever is discussed. The onset of the disease is described in detail. The four phases and clinical course of the disease are presented. The diagnostic criteria and differential diagnosis are formulated. Precise knowledge of the many manifestations of the disease will lead to early recognition of the rheumatic state.

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I esteem it a chief felicity of this country that it excels in women.

—Emerson, *Essays, Second Series: Manners.*

RHEUMATIC FEVER CASE-FINDING PROGRAM IN TWO CALIFORNIA COUNTIES*

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IN 1939, the Children's Bureau proposed that the development of a diagnostic and medical care program for children with rheumatic fever and rheumatic heart disease be initiated in a relatively small area near medical and hospital facilities. In the fall of 1940, this program was begun in Solano and Contra Costa counties and by 1941, regular diagnostic clinics were set up in the health centers at Vallejo, Pittsburg and Richmond and in the Children's Hospital of the East Bay region. These counties were chosen only because of their proximity to hospital facilities, and not because anyone suspected them of having a high incidence of rheumatic fever.

It is unfortunate that there is still no specific test for rheumatic fever such as the tuberculin, Wasserman, or agglutination tests. In the absence of a specific test, it is necessary to do a complete work-up of each case to rule out other diseases. Often it is necessary to do serial examinations for months before a diagnosis can be made. This procedure is time-consuming, and with the limited personnel in the Rheumatic Fever Program, it does not permit of a mass survey technique such as can be used in tuberculosis case-finding.

The children examined either had a previous diagnosis of rheumatic fever or were suspected of having rheumatic fever or heart disease. Therefore, the figures presented in this report and giving the percentages of normal and functional hearts found, will be small in comparison to those reported in mass surveys.

During the school year, the school physician, school nurses and teachers are alert for any rheumatic fever symptoms presented by acutely or chronically ill children. Children who present requests for rest periods or for relief from gymnasium period or for special transportation are referred to the school nurse for history investigation. If heart disease or rheumatic fever has been the reason for this request, the child is referred to the rheumatic fever clinic, unless he is under the supervision of a private physician.

From the fall of 1941 to July 1, 1944, some 442 children were examined in the four different diagnostic centers. Of this number, 235, or 56 per cent, were found to have either active rheumatic heart fever, rheumatic heart disease, or potential heart disease (meaning those children with a history of having had rheumatic fever but with no discernible heart damage). Fifty children were given deferred diagnoses and are being kept under observation in the clinics until the diagnosis is determined. One hundred and six, or 24 per cent of the children, have normal hearts and in about 33 per cent of these, a functional murmur was heard during at least one examination.

I may say here that all children are examined on at least two different occasions before a diagnosis of a normal heart or functional heart murmur is made.

Nine per cent of the children examined in this group have congenital cardiac anomalies. Seventy per cent of the children in the rheumatic group had their first attack in California, which is evidence that even though these two counties have had a tremendous increase in population because of the war industries, most of our rheumatic fever is not an imported variety from Oklahoma and

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From the Rheumatic Fever Program division of the California Department of Public Health.